is a relative dearth of data in the literature with which to guide therapy. A multicenter review of this uncommon problem is needed to better clarify the potential risks and benefits of various treatment options.

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Reply

We appreciate Dr Sternbergh's response to our article and the interesting perspective provided by his experience with two additional patients, both of whom developed deep cervical infections caused by alpha-hemolytic streptococcus within 3 weeks after carotid endarterectomy but were successfully treated by antibiotics and local wound measures without excision of their Dacron patches. Provided a satisfactory greater saphenous vein had been present in either groin, we probably would have replaced each of these patches with a vein patch simply because of the traditional concern that antibiotics, debridement, and even muscular coverage may not be sufficient to overcome bacterial contamination in a fabric arterial prosthesis. While we agree that early reoperations can be difficult, most of the morbidity in our series that was mentioned by Dr Sternbergh—the two cranial nerve injuries and a stroke related to preoperative angiography—actually occurred in patients who presented with late patch infections and required reconstruction with vein grafts.

The surgical approach that was taken for the two early postoperative infections described by Dr Sternbergh brings to mind the strategy that was introduced several years ago by Bandyk et al[1] for the management of late femoral graft biofilm infections caused by *Staphylococcus epidermidis,* another organism that generally has low clinical virulence. This strategy also emphasizes adequate debridement and coverage with viable muscle—in this case, the sartorius—but one of its important principles is replacement of all infected Dacron with nonporous polytetrafluorethylene (PTFE) in order to avoid persistent bacterial colonization in the interstices of a fabric graft. (It seems safe to say that an autogenous replacement graft would be just as appropriate as PTFE, or even more so, if one having an adequate diameter were available.) As Dr Sternbergh is the first to point out, the fact that this principle was not followed in either of his patients does not invalidate it. He made a value judgment based on the conditions as he found them, and it appears to have been correct. However, he undoubtedly followed both of these patients very closely and was prepared to excise their Dacron patches if there had been any evidence of lingering infection.

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Regarding "Management of leg ulcers in patients with rheumatoid arthritis or systemic sclerosis: the importance of concomitant arterial and venous disease"

To the Editors:

In their recent paper, Hafner et al[1] are to be commended for their thorough vascular assessment of patients with rheumatological disease who have a leg ulcer. Like ourselves,[2] they found that leg ulcers in RA often have a multifactorial etiology with little clinical evidence of vasculitis. It is important to stress, however, that serological evidence of systemic vasculitis such as hypocomplementemia or positive tests for antineutrophil cytoplasmic antibodies are usually lacking in systemic rheumatoid vasculitis and cannot be used to exclude this diagnosis. Clinical clues such as nail-fold infarcts, pericarditis, mononeuritis multiplex, or marked constitutional symptoms should be looked for. If found, patients should be referred to a rheumatologist for consideration of immunosuppressive therapy. The absence of these features, however, should prompt an assessment of large vessel function such as the authors describe. We also have anecdotal evidence of appropriate vascular intervention leading to ulcer healing in RA and support their call for a formal study of the effectiveness of surgical intervention in these patients when evidence of systemic vasculitis is absent.

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Reply

We acknowledge Dr McRorie's valuable comments in response to our publication on leg ulcers in patients with rheumatoid arthritis or systemic sclerosis. Obviously, we agree that patients with clinical signs of systemic vasculitis should be referred to a rheumatologist for further assessment and, where appropriate, for immunosuppressive therapy. Among the nine patients with rheumatoid arthritis and leg ulcers presented in our paper, none had signs of nail-fold infarcts, pericarditis, mononeuritis multiplex, renal involvement, or other symptoms of systemic vasculitis. Several among them (4/9) received additional immunosuppressive therapy for their ulcers, without improvement. It was remarkable that some of the patients (5/9) had never been subjected to vascular assessment. Often the initial cause for leg ulceration in rheumatoid arthritis remains unclear and must be left to

speculation (ie, as to whether it is due to minor trauma in atrophic skin, cutaneous small-vessel vasculitis, systemic vasculitis, or pyoderma gangrenosum).

At any rate, once the ulcer is established, impaired macrocirculation—arterial and/or venous—may greatly influence the lack of healing. In accordance with Dr McRorie we believe that a multidisciplinary approach should be encouraged for patients with rheumatological disease who have leg ulcers. A prospective study should be undertaken to investigate the role of concomitant arterial and venous disease and the effectiveness of vascular surgery in the treatment of these patients.

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Regarding "Comparing patency rates between external iliac and common iliac stents"

To the Editors:

In the article by Lee and colleagues (J Vasc Surg 2000;31:889-94), the authors have tried to test the hypothesis that external iliac and common iliac artery stents may have different long-term patency based on their anatomic differences. This is an important question that remains unresolved, and the authors are to be commended for their effort.

However in our view, the article lacks vital information to draw any valid conclusions, basically because essential data to ascertain that both groups are comparable are not provided.

It is stated in the article that "patients who had a failed attempt at stent placement were not captured in the radiologic database and could not be included in the analysis." This omission is hardly acceptable when comparing patency rates. We believe that operative failures are an integral part of patency and their exclusion is misleading.

The reporting standards for lower extremity arterial endovascular procedures[1] point out that "to allow comparison of groups of patients (. . .) objective hemodynamic test results must be provided. Doppler pressure measurements in the form of resting ankle brachial indexes are a minimum requirement." In this regard, the authors do not provide any preprocedural or postprocedural hemodynamic information from patients' limbs or from stenoses treated in spite of defining what a hemodynamic stenosis is. It is crucial to know the functional significance of these stenoses in order to make a fair comparison. Unfortunately, this information is not available in the article.

The same problem is evident when the issue of indications for reintervention is addressed. A hemodynamically significant stenosis is defined by the authors, but no data are given about what number of patients or segments needed a reintervention and what the hemodynamic status of those reintervened limbs was. So it appears that anatomic rather than hemodynamic and clinical criteria were mainly considered when patients were reintervened. Furthermore, we are puzzled by the inclusion of eight patients with 0 clinical category, without giving any justification in six of them, apparently "after consultation with the attending vascular surgeon."

Anatomic patency is just a part of the assessment of lower extremity revascularization, and obviously it is important to make sure how long any arterial intervention remains patent. The main objective, however, of any arterial intervention is to improve the hemodynamic and functional status of the limb, and this has to be evaluated by limb pressure changes or other objective criteria.

We are concerned with the publication of these data quoting extraordinarily high patency rates with major flaws in methodology. We believe that it is important to dismiss articles without appropriate methodology because they mislead rather than enlighten our understanding of clinical problems.

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Reply

The comments made by Drs Luján, Criado, Izquierdo, and Puras are much appreciated and only point to the complex nature determining iliac artery stent patency and overall outcomes. Our retrospective study was originally borne out of a disagreement between the interventional radiology and vascular surgery departments regarding the long-term patency rates and overall efficacy of external iliac artery stents. The bias in the vascular surgery department had been that external iliac artery stents had poor patency rates and did little to affect outcome. However, after a search of the literature, little information could be found regarding stents placed in the external iliac artery. The only information available was author reports examining risk factors for iliac artery stent failure. In these multivariate analyses, placement of a stent in the external iliac did not increase the risk for early stent failure. However, concerns of inadequate patient numbers (type II error) were always raised as a caveat in the discussion. To our knowledge, our manuscript is the first description of overall patency in the external iliac artery with a direct comparison to the patency of the common iliac artery.

We disagree on several points brought up by Drs Luján and colleagues. We are unclear about what is meant by the "lack of essential data to ascertain that both groups are comparable are not provided. . ." We used the recommended standards for reports dealing with lower extremity ischemia: the revised version[1] to compare levels of ischemia and to grade known risk factors within groups of patients. This information was clearly shown in tables within our manuscript.

The omission of failed attempts at stent placement is a weakness of our study, and we wish we could report those data in our manuscript. However, several papers have shown that initial success rates should be high at around 98% to 100%, so failing to identify the patients that were unsuccessfully stented probably does not affect the overall conclusions of our study.

Dr Luján and colleagues also mention our failure to include hemodynamic criteria in our manuscript. However, by reporting "anatomic patency," we did meet the recommended standards[1] for reporting patency rates, that is, "patency rates [should be] based on objective findings, like arteriography, duplex ultrasound, or magnetic resonance imaging."

Nevertheless, the inclusion of hemodynamic information is an interesting one but still does not completely answer the ultimate question: Do external iliac artery stents improve overall outcomes? Although hemodynamic data are other indicators of stent patency, we feel more information regarding stent success